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Expression of Anger in Depressed Adolescents: The Role of the Family Environment

Jennifer Jackson · Peter Kuppens · Lisa B. Sheeber · Nicholas B. Allen

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Abstract The expression of anger is considered to be abnormal in depression, yet its role is only poorly understood. In the present study we sought to clarify this role by examining the moderating influence of the family environment on overall levels of anger expression and anger reactivity in depressed and non-depressed adolescents during conflictual interactions with their parents. One hundred and forty one depressed and non-depressed adolescent participants engaged in a problem-solving task with their parents during which their behavioral expression of anger and heart rate were recorded. The results demonstrate that general levels of parental anger in the family environment (as indicated by the overall level of expressed anger by the parents during the interactions) strongly moderates how depressed differ from non-depressed adolescents in terms of their anger, heart rate and reactivity. Overall, the findings suggest that in depressed adolescents anger is much less adaptively attuned to the environment, consistent with models that predict dysfunction in the regulation of anger that prevents depressed individuals responding adaptively to their social environment.

Keywords Depression · Anger · Adolescence · Parent-child interactions · Heart rate · Behavioral observation

Anger plays an important role in the experience, onset, and maintenance of depressive symptoms and disorder (Ingram et al. 2007; Sheeber et al. 2009; Wenze et al. 2006). Indeed, the sustained presence of irritability is part of the diagnostic criteria for depression in children and adolescents (American Psychiatric Association 2000). The relationship between anger and depression is complex, however, and not well understood. Findings regarding the experience of anger in depressed and non-depressed persons have been inconsistent, with depression being associated with both greater and lesser average levels of anger, as well with both greater and lesser reactivity to anger eliciting events (e.g., Brody et al. 1999; Harmon-Jones et al. 2002; Luutonen 2007; Newman et al. 1999; Painuly et al. 2007; Sigmon and Nelson-Gray 1992).

Consideration of the social environment, particularly the family environment, may help to shed light on the inconsistencies in the relationship between depression and the experience and expression of anger. In particular, the emotional climate of the family—such as the overall level of expressed anger by the parents—may moderate an adolescent's expression of anger. Based on recent thinking about the broader social function and adaptive value of depressive behaviors, we present hypotheses regarding how the family environment may moderate differences between depressed and non-depressed youth in both their overall level of anger and in the likelihood of reacting angrily to

J. Jackson · P. Kuppens · N. B. Allen (✉)
ORYGEN Youth Health Research Centre and Department of Psychological Sciences, University of Melbourne, Victoria 3010, Australia
e-mail: nba@unimelb.edu.au

P. Kuppens
University of Leuven, Leuven, Belgium

L. B. Sheeber
Oregon Research Institute, Eugene OR, USA

specific eliciting events. We then present empirical evidence about how the overall level of parental aggression differentially relates to adolescent anger during conflictual interactions with their parents as a function of depressive status. We examine two key indices of adolescent anger during the course of such parent-adolescent interactions: 1) overall levels of adolescent anger; and 2) angry behavior in reaction to angry parental behavior (i.e., anger reactivity).

The Moderating Role of the Family Environment in Overall Levels of Adolescent Anger

Parents and children tend to mirror each other's affective behavior during the course of conflict (Forgatch 1989; Kobak et al. 1993; Sheeber et al. 2000). Given this, adolescents whose parents display high levels of anger would be expected to do the same. However, based on evolutionary theories regarding the function of depression, we postulate that the association between parental and adolescent anger is likely to be different for depressed and non-depressed adolescents. Evolutionary theories of depression suggest that depressed states evolved as adaptations to reduce risk in contexts in which individuals perceive themselves to be subject to attack as a function of lowered social status (Price and Solomon 1987; Gilbert 1989, 1992) or in which they are vulnerable to subsequent loss of status (Allen and Badcock 2003). In these situations, individuals tend to behave submissively (Gilbert 2000) and to resort to interpersonal behaviors with more certain and less variable outcomes (Allen and Badcock 2003). These behaviors are adaptive because it is not in a subordinate's interest to instigate or escalate conflicts, or make claims on resources they cannot win or defend.

These changes in social perception and behavior during depression (i.e., submissive behavior and less risky social investments) imply a potential mechanism by which social environments, including the family environment, may influence the experience of anger differentially for depressed and non-depressed adolescents. In family environments where parents express higher levels of anger and hostility, depressed, relative to non-depressed, individuals would be predicted to *suppress* anger as a means to decrease social risk and avoid conflict. Conversely, in family environments characterized by relatively lower levels of parental anger, depressed adolescents would be expected to express more anger than non-depressed adolescents. This is predicted on the basis that depression is associated with increased feelings of anger (Wenze et al. 2006), and this anger may be expressed more frequently in family environments in which adolescents feel safer and in which conflict is more likely to be resolved successfully. Accordingly, we hypothesize that although parental anger is associated with higher levels of adolescent anger overall, depressed adolescents will display relatively less anger in families with high levels of expressed parental anger, and relatively more anger in families with low expressed parental anger, when compared to non-depressed adolescents.

The Moderating Role of the Family Environment on the Adolescent's Anger Reactivity

The family environment may play an important role in moderating the adolescent's reactions to their parents' angry behaviors (i.e., anger reactivity). Importantly, this influence may differ for depressed and non-depressed youth. In general, evidence suggests that it is normative for children to more likely express their emotions in environments that are responsive and predictable (Eisenberg et al. 1998; Morris et al. 2007). Thus, in less aggressive family climates, non-depressed adolescents may feel free to reciprocate and express their anger, whereas such reciprocation may be inhibited in more aggressive climates given the potential negative consequences of aggressive reciprocation.

Yet, the concept of "emotion context insensitivity" helps to explain how the family environment may differently moderate anger reactivity in depressed youth. Evidence suggests that depressed persons are less responsive to both positive and negative stimuli, a characteristic referred to as emotion context insensitivity (ECI; Bylsma et al. 2008; Rottenberg et al. 2005). The ECI hypothesis is based on an evolutionary model that suggests that depressed states promote a defensive disengagement from one's environment (Nesse 2000). Depressive symptoms such as a loss of interest in the environment are thought to prevent depressed individuals from taking ill-considered actions (Rottenberg et al. 2005). The ECI hypothesis can, therefore, be likened to the social rank theory and social risk hypothesis (Allen and Badcock 2003; Price and Solomon 1987; Gilbert 1992) in suggesting that depressed states encourage disengagement from one's environment so as to reduce the likelihood of behaviors that may result in social defeat or rejection. Therefore, based on the ECI model, we predict that the responses of depressed youth to parental angry behavior may be less influenced by the environmental context (i.e., their family environment) than are those of non-depressed youth. The interaction between depressive status and environmental attunement and context may therefore explain the inconsistent findings with regard to between group differences in anger reactivity.

The Present Study

The aim of the present study was to better understand how the experience of anger differs as a function of adolescent depressive status and levels of parental angry behavior. The study design has a number of strengths. First, because research on emotional reactivity is enhanced by using idiographic stimuli that capture the type of emotion-eliciting events encountered in daily life (Rottenberg et al. 2005), parental and adolescent anger were assessed during parent-adolescent problem-solving interactions, which elicit personally relevant conflict behavior.

Second, adolescent anger was assessed using two objective indices, observational coding of angry behavior and heart rate, both of which capture dynamic aspects of emotional responding as they occur in real-time. Moreover, behavioral and physiological measures provide complementary information. Behavioral observations tap overt expression of anger, while physiological data can potentially reveal instances of emotional processing not otherwise observed due to behavioral inhibition of emotional expression (Gross 1998; Gross and Levenson 1993, 1997). In terms of physiology, we focused on heart rate as an indicator of anger arousal. The stress response, of which anger can be considered a part, generally involves a significant heart rate increase (Cacioppo et al. 1993; Haddy and Clover 2001). This acceleration of cardiac activity likely reflects anger's close association with the "fight" response system (Frijda 1986), which requires quick and efficient energy mobilisation (Levenson 1992). Hence, we anticipate that heart rate will increase as a function of anger concordanct with the increases in behavioral expression of anger. Nonetheless, physiological processes, including heart rate, are multiply determined such that there is not a one-to-one relationship between behavioural expressions of anger and increased heart rate. As such, examining both physiology and behavior is advantageous in that parallel findings across these indicators will enable stronger inferences to be drawn regarding the meaning of between group differences, while places where the findings diverge may provide insight into the disparate functions of these different components of emotion.

Our predictions lay out how the family environment may differentially affect the experience of anger in depressed and non-depressed adolescents. We hypothesize that in family environments characterized by higher levels of expressed parent anger, depressed adolescents will display less angry behavior and a slower average heart rate than will non-depressed adolescents. In contrast, in less angry family climates, depressed adolescents will display more angry behaviors, and a faster average heart rate than non-depressed adolescents. Furthermore, depressed adolescents will show a consistently low level of behavioral and physiological reactivity to their parents' angry behaviors, irrespective of the overall level of parental anger in the family. In contrast, non-depressed adolescents will show higher levels of anger reactivity in less angry family environments and lower levels of reactivity in more angry environments.

Method

Participants

From an initial sample of 152 adolescents, 11 were excluded for the present study due to missing physiological data, and another 45 were excluded because only one parent attended the laboratory interaction session. The remaining sample consisted of 96 participants, aged 14–18 (Mean=16 years; 37 boys), and their parents. To be included in the investigation, adolescenthad to meet research criteria for placement in one of two groups (Depressed, n=43, of which 17 boys; or Healthy, n=53, of which 20 were boys; gender and depressive status were unrelated, $\chi^2(1)=0.03$, $p=0.86$). Depressed adolescents met DSM IV (American Psychiatric Association 2000) diagnostic criteria for a current unipolar depressive disorder. Consistent with DSM guidelines for establishing the offset of depressive episodes, a diagnosis was considered current if it was ongoing or had an offset within 2 months preceding the diagnostic interview (American Psychiatric Association 2000); using this definition, two of the depressed adolescents were in partial remission at the time of the assessment. Healthy adolescents had no lifetime history of psychopathology or mental health treatment. Adolescents were excluded if they evidenced comorbid externalizing or substance dependence disorders or were taking medications with known cardiac effects. Eight participants reported taking other forms of medication (all from the depressed group), and 17 were undergoing treatment during the time of the study (of which 16 were from the depression group).

Recruitment and Assessment Procedures

Families were recruited using a two-gate procedure consisting of an in-school screening and an in-home diagnostic interview. In order to facilitate recruitment of a representative sample of students, we used a combined passive parental consent and active student assent protocol for the school screening (Biglan and Ary 1990; Severson and Ary 1983). A letter describing the purpose, procedures, risks, and benefits of completing the questionnaire was mailed to the parents of each student 2 weeks before the screening. Parents who wished to decline participation returned a stamped and pre-addressed postcard to the school. Addi-

tionally, on the day of the assessment, this information was provided verbally and in writing to the students, who signed the written information form if they chose to assent. Students who did not assent, whose parents declined permission, or for whom letters to parents were returned as undeliverable, were excluded. Active parent consent and adolescent assent for the full assessment were obtained prior to the diagnostic interview.

School screening Students (N=4182) from area high schools completed the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff 1977) and a demographic form during class. Approximately 70% of eligible students participated, 12% declined or had parents decline their participation and 18% were absent or off campus on the day of the assessment. CES-D cut-off scores for sample selection were based on the distribution of scores obtained in an earlier screening of high school students in the same area (N=4495; Sheeber et al. 2007). Relatively high scores (≥31 for males and ≥38 for females) were selected to maximize the positive predictive power to identify adolescents experiencing depressive disorder. The pool for the healthy group was defined as students not more than ½ SD above the mean of the earlier sample (<21 for males and <24 for females).

Diagnostic assessment Interviewers conducted the Schedule of Affective Disorders and Schizophrenia-Children's Version (K-SADS, Orvaschel and Puig-Antich 1994) with adolescents who had elevated CES-D scores. Subsequent to the interviews, families of adolescents who met criteria for MDD were invited to participate in the lab-based assessment. After each adolescent in the depressed group completed the lab assessment, a healthy, demographically matched comparison participant was recruited from the pool of students who scored within the normal range on the CES-D. Of families invited to participate, approximately 26% declined. Rates of decline did not vary as a function of pre-interview group status (i.e., elevated or healthy CES-D score), age, or race, though more males than females declined (31.6% vs. 23%), χ^2 (1,n=498)=4.57, p<0.05. Of adolescents with elevated CES-D scores who participated in the interview, 38% met criteria for MDD. Of adolescents with CES-D scores in the healthy range, approximately 76% met criteria for inclusion.

Lab assessment Families who met criteria for the investigation after the diagnostic interview were invited to participate in the lab assessment. Approximately 4% of families declined. The decline rate did not vary as a function of group status, age, race, or gender. In approximately 93% of two-parent families, both parents participated. Participants were instructed to abstain from alcohol and illicit drugs on the day of the assessment. Compliance with this instruction was confirmed on the day of the assessment via self report. The lab assessment included an 18-minute parent-adolescent Problem-Solving Interaction (PSI) in which families were asked to discuss and resolve two areas of conflict identified based on parent and adolescent responses on the Issues Checklist (Prinz et al. 1979). The PSI was videotaped for behavioural coding. Adolescent electrocardiograph activity was measured during the interaction.

Measures

Depression Screener

The CES-D is a widely used self-report measure of depressive symptoms that has acceptable psychometric properties for use with adolescents (e.g., Roberts et al. 1990; Radloff 1977). It has a well-established record of use as a screener in adolescent samples (e.g. Asarnow et al. 2005; Sheeber et al. 2007).

Diagnostic Interview

The K-SADS was conducted with the adolescents in order to obtain current and lifetime diagnoses. Interviewers, who were bachelor and masters level research staff, participated in a rigorous training program and demonstrated agreement with a senior interviewer (κ≥0.80) on at least two interviews before conducting independent interviews. All interview-derived diagnoses were confirmed by supervisors who reviewed both item endorsement and interviewers' notes. Questions regarding the accuracy of diagnoses were resolved based upon discussion with the interviewer and review of the audiotaped interview as needed. Based on a random selection of 20% of the interviews, inter-rater reliability was κ=0.94.

Behavioral Coding

The Living in Family Environments Coding System (LIFE; Hops et al. 1995) was used to code the adolescent's and each parent's behavior (i.e., mothers and fathers coded separately) during over the entire PSI. The LIFE is an event-based, microanalytic coding system in which a new code is entered each time there is a change in a participant's verbal content or affective behavior. Each entry is comprised of several components which identify the: a) target (i.e. whose behavior is being coded); b) verbal content; and c) nonverbal (or para-verbal) affect. Data analysis is done at the level of constructs, which are operationalized as particular combinations of content and affect codes. Observers, blind to diagnostic status, coded family members' nonverbal affect and verbal content in real time. A construct reflecting angry behavior was derived from

individual affect and content codes. Angry behavior included aggressive (e.g., raised voice; clenched teeth) or contemptuous (e.g., eye rolling; sneering) nonverbal behavior and cruel (e.g., mocking; insults; threats) or provoking (e.g., taunts; dares) statements. Approximately 25% of the videos were coded by an additional observer for reliability. Inter-rater reliability (κ) for angry behavior equaled 0.73. The validity of the LIFE system as a measure of family processes has been established in prior studies of adolescent depression (e.g., Katz and Hunter 2007; Sheeber et al. 2007).

Physiological Recording

Heart rate data were acquired using software and equipment from the James Long Company (www.jameslong.net). The ECG signals were recorded using Ag-AgCl electrodes. To record the ECG signal, a three-lead system was used to maximize the r-wave amplitude, and to minimize movement artifact and t-wave amplitude. The positive electrode was placed directly under the left armpit about 4–6 inches down, at heart elevation. The negative electrode was placed directly under the right armpit at the same elevation. The ground electrode was placed on the sternum, halfway between the positive and negative electrodes. The ECG signals were input to an isolated bioelectric amplifier, with a gain of 250 and bandpass of frequencies between 0.1 and 1000 Hz. Heart rate was calculated based on the time (in milliseconds) between successive r-waves (R-R intervals) on the ECG. The ECGRWAVE program identified r-waves from the ECG signal with an automated, multiple-pass, self-scaling algorithm. These signals were then visually inspected. Missed or misrepresented r-waves were manually corrected and sections of movement, noise, or flat line artifact were removed.

Data Analysis

The data were analyzed using autocorrelation-crosscorrelation regression models in a multilevel framework. The multilevel regression framework takes into account dependencies in the data resulting from the nested structure (second-by-second observations nested in individual participants), and separates within from between person variance (Bryk and Raudenbush 1992; Snijders and Bosker 1999). The autocorrelation-crosscorrelation approach enables examination of the influence of one variable on *change* in a second variable, and is the closest means by which to infer a causal relationship using multivariate time series data (see Gottman 1990; Granger 1969).

Multi-level regression models were estimated separately for each of the dependent variables (i.e., adolescent angry behavior and heart rate). To model the physiological data, normal multilevel regression models were used. However, to model the adolescents' behavioral data, a logistic transformation function was added (resulting in logistic multilevel regression) to accommodate for their binary nature.

At level one of the models, the dependent variable (e.g. the adolescent's anger display or heart rate) was predicted firstly by an *intercept.* The intercept reflects the average level of expressed anger or heart rate, which was allowed to vary across participants (due to the multilevel modelling approach). The intercept is thus relevant to the research questions relating to average levels of anger in the adolescent. Additionally, the dependent variable was also predicted by maternal and paternal anger displayed 5 s earlier (the cross correlation) while controlling for the dependent variable 5 s earlier (the autocorrelation). All predictor variables were group-mean centered (Enders and Tofighi 2007). The *slopes* of the predictor variables also varied across individuals and reflect the adolescents' behavioral and physiological reactivity in response to their parents' display of anger. A 5 s lag has been used successfully in our prior work with these measures (Allen et al. 2010; Kuppens et al. 2010).

At level two of the models, both the person-specific intercept and slope values were modeled as a function of adolescent depression (binary coded as a 1 for depressed, and 0 for non-depressed), the average level of observed parental anger during the interaction (calculated as the average level of angry behaviours demonstrated by the adolescent's parents throughout all interactions, collapsed across mothers and fathers and centered; *Mean* before centering=0.07, *SD*=0.07) and the interaction of depression and parental anger. Their effects on the *intercept* reflect how these factors relate to the adolescents overall level of anger during the interaction. The effect of these factors on the *slope* reflects how these factors relate to adolescent anger reactivity (see Appendix for more detail on the used model).

Results

Preliminary Analyses

As the addition of level 1 predictor variables changes the interpretation of the intercept, a preliminary analysis was performed to determine whether levels of overall anger expression and heart rate were significantly different between depressed and non-depressed adolescents, without additionally including predictors at level 1 of the model. In two random intercept models, adolescent angry behavior and heart rate were predicted by a random intercept at level one, which at level two, was modeled as a function of depression only. The results of these analysis indicated that depressed youth expressed more anger on average

than non-depressed adolescents, B=−2.123, SE=0.169, p=0.000, and had a higher heart rate, B=75.206, SE=1.178, p=.000, during the interactions (see also Sheeber et al. 2009; Allen et al. 2010; Byrne et al. 2010). Following this, the main analyses described above were conducted. The findings for anger behavior and heart rate are reported in Tables 1 and 2, respectively.

In addition, for comparison, unconditional models were estimated predicting adolescent anger and heart rate (including only a random intercept term without level 2 predictors). For adolescent anger, the average intercept value equalled −1.70, T-ratio=−10.71, p<0.001; variance=2.42. For heart rate, the average intercept equalled 77.80, T-ratio=78.20, p<0.001; variance=95.98.

Average Adolescent Anger and Heart Rate (Intercept)

Adolescent Anger Behavior

There was a significant main effect of overall parental anger, collapsed across mothers and fathers, on the average level of adolescent angry behaviors (Table 1). Adolescents whose parents displayed greater levels of angry behavior overall, displayed more angry behavior themselves. This effect was significant for both depressed, $\chi^2(1)=6.73$, p=0.009, and non-depressed, $\chi^2(1)=20.59$, p=.000, adolescents. The interaction between depression and parental anger was also significant, however, indicating that differences between depressed and non-depressed adolescents varied as a function of levels of parental angry behavior. As can be seen in Fig. 1a, in families with low parental expressed anger, depressed adolescents exhibited significantly more angry behavior than did non-depressed adolescents, $\chi^2(1)=12.99$, p=0.0006, while no between group differences emerged in families with high parental expressed anger, $\chi^2(1)=0.10$, p>0.500.[1]

[1] It should be noted that in the full analysis reported in Table 1, the effect of depression on average levels of adolescent anger became only marginally significant in contrast to the previously reported preliminary random-intercept model. Follow-up analyses revealed that this change in significance was *not* due to the inclusion of parental anger and the interaction term in predicting the intercept. Random intercept analyses which included depression, anger in the family environment, and their interaction, showed that all three variables significantly contributed to the intercept (Depression: B=0.461, SE=0.204, p=0.025; parental anger: B=0.14.97, SE=2.332, p=0.000; interaction: B=−7.385, SE=2.845, p=0.011), demonstrating that the effect of depression on average adolescent anger was not mediated by these other factors. Instead, the change in significance was a result of the inclusion of the level 1 group-mean centered predictor variables (autocorrelation and cross-correlations) and their level 2 moderators (Snijders and Bosker 1999). It can therefore be safely concluded that depressed adolescents indeed displayed more anger on average compared to non-depressed adolescents in the present study, despite the lack of significance of depression reported in Table 1.

Adolescent Heart Rate

The results regarding the intercept in Table 2 again show that depressed adolescents had a higher heart rate throughout the interactions when compared to non-depressed adolescents, regardless of family climate. There was, however, again a significant interaction between depression and family climate, which is graphically portrayed in Fig. 1b. In families with low parental expressed anger depressed adolescents had a higher average heart rate than non-depressed adolescents, $\chi^2(1)=12.34$, p=0.001, but this was not the case in families with higher parental expressed anger, $\chi^2(1)=0.00$, p>0.500. Moreover, although aggressive family climate was marginally associated with increased levels of heart rate overall (see Table 2), the results presented in Fig. 1b show that this trend only held for non-depressed adolescents, $\chi^2(1)=2.96$, p=0.081, whereas for depressed adolescents this difference was non-significant, $\chi^2(1)=1.54$, p=0.212.

Adolescent Anger Reactivity (Slope)

Adolescent Anger Behavior

In response to displays of anger by the mother, there was a significant main effect of overall parental anger on adolescent behavioral anger reactivity whereby adolescents in environments characterized by lower levels of parental angry behavior were more reactive to parental anger (see results with respect to slopes in Table 1). However, this main effect was moderated by depressive status as displayed in Fig. 2a. As can be seen, the effect of overall parental anger occurred predominantly for non-depressed adolescents, $\chi^2(1)=6.91$, p=0.008. Overall levels of parental anger did not significantly influence the way in which depressed adolescents responded to maternal anger, $\chi^2(1)=0.18$, p>0.500.

Hence, within less angry family climates, non-depressed adolescents showed greater behavioral reactivity than depressed adolescents, $\chi^2(1)=4.33$, p=0.035, whereas no such difference was found in more angry families, $\chi^2(1)=0.68$, p>0.500. No significant main effects, interaction effects, or significant contrasts were found for behavioral adolescent anger reactivity in response to paternal angry behavior (see Table 2, and Fig. 2b).

Adolescent Heart Rate

Physiologically, a similar pattern emerged. There was a significant main effect of overall parental anger as well as a significant interaction effect with depression on adolescent heart rate reactivity in response to mothers' angry behaviour (Table 2). As can be seen in Fig. 2c, the heart rate of

Table 1 Results from logistic multilevel analysis predicting adolescent angry behaviour at time t by a random intercept, t-5 s lagged adolescent angry behaviour (not listed) and t-5 s angry behaviour by the Mother and the Father, as a function of depression, overall levels of expressed parent anger in the family environment (overall anger) and the interaction of depression and parental anger (interaction)

Level 1 term (*Var*[a])	Coefficient (β)	Standard Error (*SE*)	*p*-value
Intercept (1.81, $p<0.001$)			
Intercept (η_0^0)	−1.663	0.106	0.000
Depression (η_1^0)	0.235	0.143	0.103
Overall Anger (η_2^0)	9.707	2.139	0.000
Interaction (η_3^0)	−5.616	2.657	0.036
Slope Mother (0.34, $p<0.001$)			
Intercept (η_0^2)	0.213	0.039	0.000
Depression (η_1^2)	−0.073	0.067	0.281
Overall Anger (η_2^2)	−1.745	0.664	0.010
Interaction (η_3^2)	2.025	0.933	0.032
Slope Father (0.46, $p<0.001$)			
Intercept (η_0^3)	0.126	0.052	0.017
Depression (η_1^3)	−0.004	0.081	0.957
Overall Anger (η_2^3)	0.319	0.870	0.714
;Interaction (η_3^3)	−0.408	0.969	0.674

See appendix for model equations. [a] Between parentheses is the variance of the corresponding random effect with *p*-value

non-depressed adolescents increased in response to mothers' anger in more angry family environments, while in less angry environments, it decreased, $\chi^2(1)=7.06$, $p=0.008$). There were no differences for depressed adolescents, $\chi^2(1)=1.53$, $p=0.213$. Within less angry family environments, contrasts furthermore revealed significant differences between depressed and non-depressed adolescents, $\chi^2(1)=6.16$, *v* $p=0.013$. Non-depressed adolescents showed heart rate decrease, while depressed youth experienced heart rate increase. In more angry environments, group differences were not significant, $\chi^2(1)=2.02$, $p=0.152$.

Interestingly, despite the absence of behavioral differences, there was a main effect of depression on the physiological reactivity of adolescents in response to fathers' angry behaviors. Depressed adolescents consistently showed heart rate increase while non-depressed adolescents showed decrease in response to fathers' anger, regardless of the affective climate of the family environment. Contrasts revealed that group differences in this pattern of reactivity was more pronounced within angrier family environments, $\chi^2(1)=6.28$, $p=0.012$, as opposed to less angry environments, $\chi^2(1)=2.22$, $p=0.132$. These findings are graphically represented in Fig. 2d.

The same series of analyses, including gender (dummy coded with 1=male and 0=female) as a level-2 predictor above the other predictors, was run as well. The results and conclusions remained the same, save from additionally showing that girls on average expressed more anger during the interactions than boys, B=−0.38; *T*-ratio=−2.99, $p=0.004$, and had marginally significant higher heart rate

Table 2 Results from multilevel analysis predicting adolescent heart rate at time t by a random intercept, t-5 s lagged adolescent heart rate (not listed) and t-5 s angry behaviour by the Mother and the Father, as a function of depression, overall levels of expressed parent anger in the family environment (overall anger) and the interaction of depression and parental anger (interaction)

Level 1 term (*Var*[a])	Coefficient (β)	Standard Error (*SE*)	*p*-value
Intercept (88.23, $p<0.001$)			
Intercept (η_0^0)	76.456	1.314	0.000
Depression (η_1^0)	4.782	1.970	0.017
Overall Anger (η_2^0)	41.795	24.277	0.088
Interaction (η_3^0)	−66.652	31.462	0.037
Slope Mother (1.90, $p<0.001$)			
Intercept (η_0^2)	−0.265	0.219	0.229
Depression (η_1^2)	0.371	0.361	0.308
Overall Anger (η_2^2)	11.279	4.243	0.010
Interaction (η_3^2)	−14.141	4.835	0.005
Slope Father (2.21, $p<0.001$)			
Intercept (η_0^3)	−0.440	0.254	0.087
Depression (η_1^3)	0.929	0.448	0.041
Overall Anger (η_2^3)	−0.436	3.688	0.907
Interaction (η_3^3)	−2.134	4.642	0.653

See appendix for model equations. [a] Between parentheses is the variance of the corresponding random effect with *p*-value

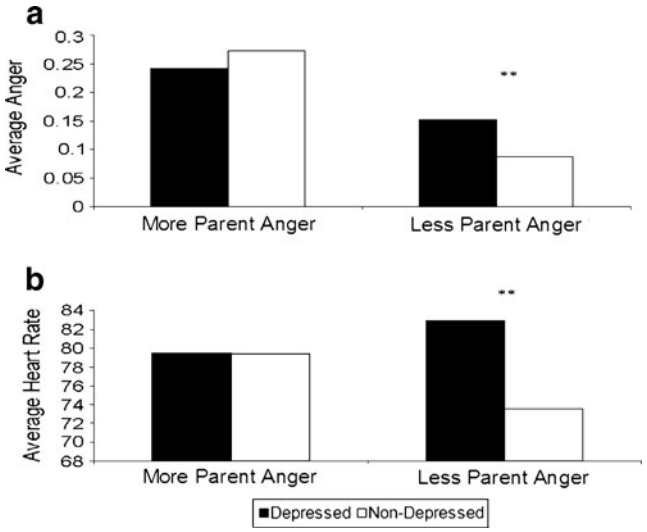

Fig. 1 Predicted mean scores for depressed and non-depressed adolescents from families high and low (+/−1 SD) in expressed parental anger of **a** average behavioural expression of anger, and **b** average heart rate. The asterisks (**) indicate a significant contrast at the level of $p=0.001$

overall, B=−3.50; *T*-ratio=−1.68, $p=0.095$. There were no significant gender differences in anger reactivity (in response to mother: B=−0.08; *T*-ratio=−1.22, $p=0.23$; in response to father: B=−0.09; *T*-ratio=−1.12, $p=0.27$) or in heart rate reactivity (in response to mother: B=−0.08; *T*-ratio=−0.212, $p=0.83$; in response to father: B=0.50; *T*-ratio=1.20, $p=0.23$).

Discussion

Depression is most typically associated dysphoric mood, yet research suggests that the emotional dysfunction evident in depression also extends to the domain of anger. Previous research has yielded inconsistencies with regard to differences in the nature and mechanism of anger regulation and expression between depressed and nondepressed persons.

In the present paper, we wanted to shed light on this issue by examining how the family climate may differentially moderate anger in depressed and non-depressed adolescents during conflictual interactions with their parents. The overall pattern that emerged (see Figures), illustrates that non-depressed adolescents' levels of angry behavior and physiology mirror that of their parents' behavior, whereas this is significantly less so (in terms of behavior) or even reversed (in terms of physiology) for depressed adolescents. In other words, the finding that children's regulation of anger mirrors

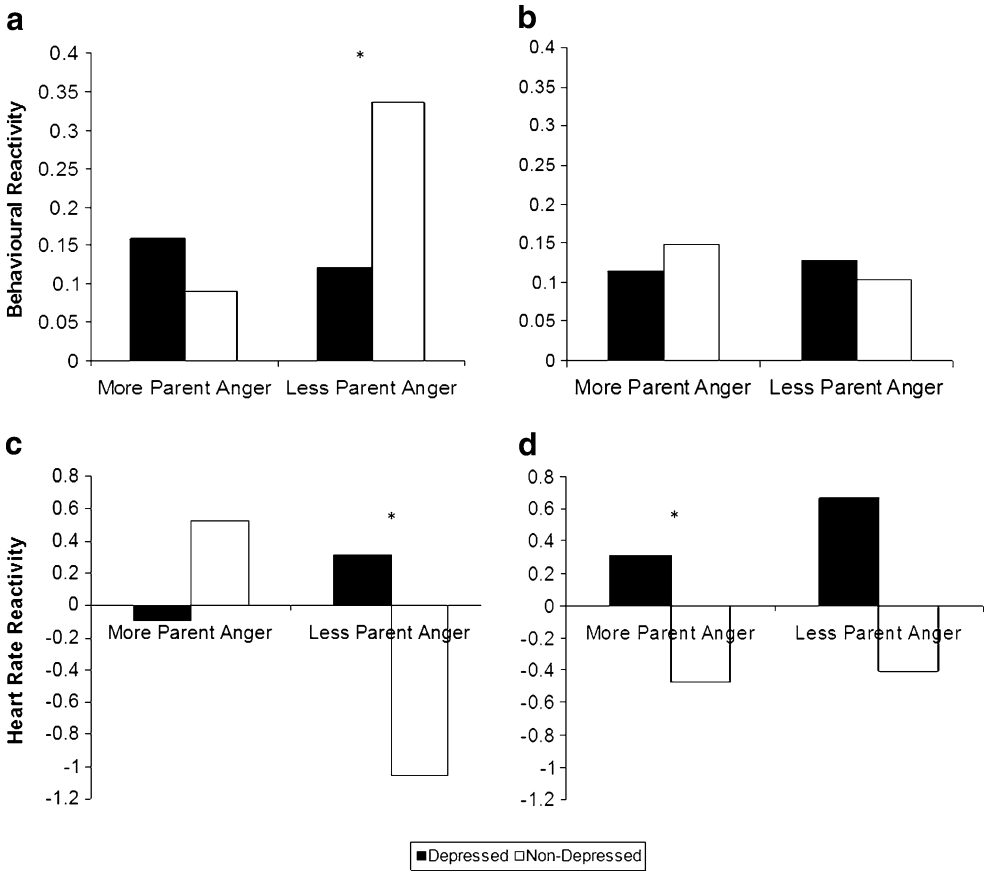

Fig. 2 Predicted mean changes in behaviour (**a**, **b**) and heart rate (**c**, **d**) in response to displays of anger by mother (**a**, **c**) and father (**b**, **d**) for depressed and non-depressed adolescents from families high and low (+/−1 SD) in expressed parental anger. The asterisk (*) refers to a significant contrasts at the level of $p=0.05$

that of their parents, as well as that parents and children tend to show a matching of affect and behavior during the course of conflict (Forgatch 1989; Kobak et al. 1993), seems to apply less to depressed than to non-depressed adolescents. As such, the findings from this study indicate that in contrast to non-depressed adolescents, depressed adolescents' anger expression is not as contingent upon usual cues in their social environment. This overall pattern was apparent in data on both heart rate and anger behavior, both in terms of overall level as well as reactivity, and expressed itself in significant interaction effects between depressed status and overall family anger expression style on these parameters (with effects sizes ranging from 0.22 to 0.28 for the reported interaction effects).

Being less in tune with their social environment when expressing anger may be particularly problematic for depressed adolescents, given that, consistent with reports that depressed persons experience greater levels of anger than non-depressed persons (Fava and Rosenbaum 1998; Ingram et al. 2007; Sheeber et al. 2000; Wenze et al. 2006), we found that depressed adolescents demonstrated both more angry behavior and higher heart rates than did non-depressed adolescents. However, we argued that because depressed mood has been hypothesised to have evolved to serve socially adaptive functions, consideration of the social context of anger would facilitate a better understanding of how the experience of anger differs between depressed and non-depressed adolescents. The findings indeed demonstrated that the emotional climate of the family significantly moderates the relationship between depression and the expression of anger. Depressed adolescents displayed higher overall levels of anger expression and heart rate in families characterized by lower levels of parental anger, whereas this difference was not present in families characterized by high levels of parental anger. In line with our hypothesis, this finding provides support for the notion that increased anger expression by depressed individuals occurs in environments that pose a lower risk of social defeat or rejection. Moreover, these findings add specificity to Joiner's (2000) theory of interpersonal conflict avoidance by highlighting boundary conditions under which such avoidance may occur. That is, while interpersonal conflict avoidance (i.e., low assertiveness, social withdrawal, avoidance and shyness) may indeed prolong and exacerbate symptoms of depression in social environments whereby the individual is at greater risk of social loss (e.g. environments where increased anger is expressed by others), in less angry social environments, it may in fact be the depressed individuals' poorer attunement to social contingencies that exacerbate their depressive symptoms.

In terms of anger reactivity to parental angry behavior, we hypothesized that non-depressed adolescents' reactivity would be moderated by the level of parental anger, such that higher reactivity would be observed in an environment of low overall levels of parental anger, where it has been conjectured that the adolescent will feel more comfortable expressing negative emotion (Morris et al. 2007), and low reactivity would be observed in families with high levels of parental anger. Our results supported this hypothesis for both behavioral anger expression and heart rate reactivity in response to displays of anger by the mother. In families with low levels of parental anger, non-depressed adolescents responded to their mothers' aggression with increases in angry behavior. Moreover, this increase in anger expression was associated with a marked slowing down of heart rate among non-depressed adolescents. Heart rate decrease is considered a strong indicator of an orienting response (Graham and Clifton 1966; Richards and Casey 1992; Turpin 1986). Such a response occurs when a situation requires "attention" and, therefore, functions to increase an individual's sensitivity to their surrounding environment (Graham and Clifton 1966). Subsequently, information intake from the environment is increased, and the individual can better adapt to the triggering stimulus (e.g., the adolescents' parents' angry behaviors during conflict). Our findings, therefore, suggest that non-depressed adolescents display a pattern of reactivity to their parents' anger that is adaptively modified by the nature of the family environment.

Interestingly, the largest heart rate decrease in non-depressed adolescents occurred in those conditions where increased behavioral reactivity was observed (i.e., in response to their mother's anger, in less angry environments). This pattern suggests that such conditions may be an optimal setting for successful negotiations of parent-adolescent conflict, in which the adolescent learns how to deal with conflict in an adaptive way (Gunlicks-Stoessel and Powers 2008). The fact that this was only found in response to the mother appears consistent with prior research; not only do parent-adolescent conflicts predominantly involve the mother (Furman and Buhrmester 1992), but the mother tends to be the target of a greater amount of expressed anger than the father during such disagreements (Davis et al. 1998).

Meanwhile, on the basis of the ECI hypothesis, which proposes environmental disengagement during depressed states, we hypothesized that anger reactivity in depressed adolescents would be relatively unaffected by levels of parental anger. Consistent with this hypothesis, depressed adolescents did not show greater behavioral anger reactivity towards their mothers in less angry family environments. Furthermore, depressed adolescents demonstrated heart rate *increase* rather than decrease in

response to their fathers' displays of anger during conflict. Heart rate increase is considered a strong indicator of negative affect, and of the stress (or fight) response during anger (Buss et al. 2005; Cacioppo et al. 1993; Haddy and Clover 2001; Levenson 1992). Furthermore, in contrast to the orienting response, this defense response facilitates disengagement from the environment, including rejection of incoming sensory information (Cook and Turpin 1997; Turpin 1986). The defense response tends to occur when the triggering stimulus is intense, unpleasant, or implies personal danger (Graham and Clifton 1966); this response, therefore, contributes to escape-mobilisation of the individual. Unlike non-depressed adolescents, depressed adolescents therefore predominantly show a physiological stress response to paternal anger that reflects defensive disengagement from that environment and the absence of a behavioural response to maternal anger.

These differences in reactivity between depressed and nondepressed adolescents suggest that it may be adaptive to modulate anger expression contingent upon both the broader environment and others' specific behaviors during conflict. Yet, environmental disengagement in situations of conflict appears to prevent this for depressed adolescents. An understanding of how depressed and non-depressed adolescents differ in their appraisal of others' anger during conflict may enable clinical interventions to encourage reappraisal strategies during conflict.

In sum, the overall pattern that emerges from these findings is one of depressed individuals' anger being poorly attuned to their environment. Although our findings confirm that depressed adolescents are characterized by higher overall levels of anger expression and associated physiology, this increased anger does not coincide with increased reactivity to putatively anger eliciting aspects of their environment. Therefore, the increased levels of anger observed in depression are not due to increased reactivity to normatively anger-provoking situations, but rather to increased frequency of anger instances that are not in response to typical anger provocations such as parental displays of anger. Moreover, whereas non-depressed adolescents' anger levels mirror parental anger expression, this is not the case for depressed adolescents, and the anger reactivity of depressed individuals appears to be less moderated by the family environment. In sum, anger reactivity of depressed seems less attuned to both the immediate or broader environment. The anger that results may be particularly maladaptive as it does not arise in response to typical anger-provoking circumstances or family environments, and therefore may not result in adaptive outcomes for the problems that anger evolved to solve.

One limitation to this study is that it does not enable us to determine the extent to which these differences have etiological significance. Future investigation comparing the experience of anger across at-risk, clinically depressed, and non-depressed individuals, or which followed at risk individuals longitudinally, would be necessary in order to draw stronger conclusions. However, we speculate that the maladaptive expression of anger by depressed adolescents over time could function to exacerbate, or contribute to increased interpersonal conflicts at future time-points. This generation of interpersonal stress could, in turn, prolong the adolescent's experience of depressive symptoms. Furthermore, the broader family environment was classified solely by the degree of expressed parental anger. The emotional climate of the family, of course, reflects multiple intra-familial processes and dynamics (Morris et al. 2007), which should be considered in future studies. For example, the role of siblings and/or levels of positive emotionality are important issues yet to be addressed. Given that fathers are noticeably underrepresented in studies of emotional socialization (Parke and McDowell 1998; Phares 1992), their inclusion in our study was an important advantage. The limitations to the presented findings should also be contrasted to other strengths such as that the generation of parent-adolescent conflict using idiographic and personally-relevant issues, providing sound ecological validity, and the use of a combination of physiological and behavioral measures.

In summary, considering the broader emotional climate of the family environment provided important clues to understanding abnormal anger regulation in depression. Although depressed adolescents were shown to be characterized by higher levels of anger expression and physiology during conflictual interactions with their parents, these anger behaviors were less likely to occur in response to the usual environmental elicitors. In all, the results suggest that anger in depression is much less adaptively attuned to the environment, suggesting a fundamental dysfunction in the regulation of anger that prevents depressed individuals responding adaptively to their social environment. These finding may have important clinical implications, especially when working with families of depressed adolescents. It may be helpful for families to understand that adolescents suffering from depression are likely to be angrier, although their expressions of anger may appear unpredictable. Understanding that these behaviors may be part of the depressive syndrome is likely to help families to understand and tolerate them better, and indeed this pattern of behavior may be an appropriate target for focused therapeutic intervention given its potential role in maintaining and exacerbating depressive conditions.

Appendix

For HR, the following multilevel model was estimated:

$$HR_{t,j} = \beta_{0j} + \beta_{1j}HR_{t-5s,j} + \beta_{2j}Mother_{t-5s,j} + \beta_{3j}Father_{t-5s,j} + \varepsilon_{t,j}$$

with

$$\beta_{0j} = \eta_0^0 + \eta_1^0 Depr_j + \eta_2^0 ParentAggr_j + \eta_3^0 Depr_j \times ParentAggr_j + \varepsilon_j^0$$
$$\beta_{1j} = \eta_0^1 + \eta_1^1 Depr_j + \eta_2^1 ParentAggr_j + \eta_3^1 Depr_j \times ParentAggr_j + \varepsilon_j^1$$
$$\beta_{2j} = \eta_0^2 + \eta_1^2 Depr_j + \eta_2^2 ParentAggr_j + \eta_3^2 Depr_j \times ParentAggr_j + \varepsilon_j^2$$
$$\beta_{3j} = \eta_0^3 + \eta_1^3 Depr_j + \eta_2^3 ParentAggr_j + \eta_3^3 Depr_j \times ParentAggr_j + \varepsilon_j^3$$

where t denotes time and j denotes the participant. At level 1 of the model, adolescents' HR at time t is predicted by a person-specific intercept, the (group-mean centered) adolescents' HR at time t-5 s ($HR_{t\text{-}5s,j}$) with a person-specific slope, (group-mean centered) maternal angry behaviour at time t-5 s ($Mother_{t\text{-}5s,j}$) with a person-specific slope, and (group-mean centered) paternal angry behaviour at time t-5 s ($Father_{t\text{-}5s,j}$) with a person-specific slope. At level 2 of the model, the person-specific intercept and slope values are predicted by a general intercept, depression (coded as 1=depressed; 0=non-depressed), the centered, overall level of parental aggression expressed throughout the interactions (*Mean*=0, *SD*=0.07), and the interaction between both. An identical but logistic regression model was estimated for anger behaviour.

References

Allen, N. B., & Badcock, P. B. T. (2003). Darwinian models of depression: a review of evolutionary accounts of mood and mood disorders. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 30*, 815–626.

Allen, N. B., Kuppens, P., & Sheeber, L. B. (2010). *Heart rate responses to parental behavior in depressed adolescents*. Manuscript submitted for publication.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Asarnow, J. R., Jaycox, L. H., Duan, N., LaBorde, A. P., Rea, M. M., Murray, P., et al. (2005). Effectiveness of a quality improvement intervention for adolescent depression in primary care clinics: a randomized controlled trial. *Journal of the American Medical Association, 293*, 311–393.

Biglan, A., & Ary, D. V. (1990). Methodological issues in research on smoking prevention. In C. S. Bell & R. Battjes (Eds.), *Prevention research: Deterring drug abuse among children and adolescents (NIDA Research Monograph Series, No. 63)* (pp. 170–195). Rockville, MD: U.S. Department of Health and Human Services.

Brody, C. L., Haaga, D. A. F., Kirk, L., & Solomon, A. (1999). Experiences of anger in people who have recovered from depression and never-depressed. *Journal of Nervous and Mental Disease, 187*, 400–405.

Bryk, A. S., & Raudenbush, S. W. (1992). *Hierarchical linear models for social and behavioural research: Applications and data analysis methods*. Newbury Park: Sage Publications.

Buss, K. A., Goldsmith, H. H., & Davidson, R. J. (2005). Cardiac reactivity is associated with changes in negative emotion in 24-month-olds. *Developmental Psychobiology, 46*, 118–132.

Bylsma, L. M., Morris, B. H., & Rottenberg, J. (2008). A meta-analysis of emotional reactivity in major depressive disorder. *Clinical Psychology Review, 28*, 676–691.

Byrne, M., Sheeber, L., Simmons, J., Davis, B., Shortt, J., Katz, L., et al. (2010). Autonomic cardiac control in depressed adolescents. *Depression and Anxiety*. doi:10.1002/da.20717. Epub ahead of print.

Cacioppo, J. T., Berntson, G. G., Laresen, J. T., Poehlmann, K. M., & Ito, T. A. (1993). The psychophysiology of emotion. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (2nd ed., pp. 173–191). New York: Guilford.

Cook, E. C., III, & Turpin, G. (1997). Differentiating orienting, startle, and defense response: The role of affect and its implications for psychopathology. In P. J. Lang, R. F. Simons, & M. Balaban (Eds.), *Attention and orienting. Sensory and motivational processes* (pp. 137–164). New Jersey: Lawrence, Erlbaum Associates Inc.

Davis, B. T., Hops, H., Alpert, A., & Sheeber, L. (1998). Child responses to parental conflict and their effect on adjustment: a study of triadic relations. *Journal of Family Psychology, 12*, 163–177.

Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry, 9*, 241–273.

Enders, C. K., & Tofighi, D. (2007). Centering predictor variables in cross-sectional multilevel models: a new look at an old issue. *Psychological Methods, 12*, 121–138.

Fava, M., & Rosenbaum, J. F. (1998). Anger attacks in depression. *Depression and Anxiety, 8*, 59–63.

Forgatch, M. S. (1989). Patterns and outcome in family problem solving: the disrupting effect of negative emotion. *Journal of Marriage and the Family, 51*, 115–124.

Frijda, N. H. (1986). *The emotions*. Cambridge: Cambridge University Press.

Furman, W., & Buhrmester, D. (1992). Age and sex differences in perceptions of networks of personal relationships. *Child Development, 63*, 103–115.

Gilbert, P. (1989). *Human nature and suffering*. Hove: Lawrence Erlbaum.

Gilbert, P. (1992). *Depression: The evolution of powerlessness*. Hove: Lawrence Erlbaum.

Gilbert, P. (2000). The relationship of shame, social anxiety and depression: the role of the evaluation of social rank. *Clinical Psychology and Psychotherapy, 7*, 174–189.

Gottman, J. M. (1990). Time-series analysis applied to physiological data. In J. T. Cacioppo & L. G. Tassinary (Eds.), *Principles of psychophysiology: Physical, social and inferential elements* (pp. 754–774). New York: Cambridge University Press.

Graham, F. K., & Clifton, R. K. (1966). Heart-rate change as a component of the orienting response. *Psychological Bulletin, 65*, 305–320.

Granger, C. W. J. (1969). Investigating causal relations by econometric models and cross-spectral methods. *Econometrica, 37*, 424–438.

Gross, J. J. (1998). The emerging field of emotion regulation: an integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: the acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*, 95–103.

Gunlicks-Stoessel, M. L., & Powers, S. I. (2008). Adolescents' emotional experiences of mother–adolescent conflict predict internalizing and externalizing symptoms. *Journal of Research on Adolescence, 18*, 621–642.

Haddy, R. I., & Clover, R. D. (2001). The biological processes in psychological stress. *Families Systems and Health, 19*, 291–302.

Harmon-Jones, E., Abramson, L. Y., Sigelman, J., Bohlig, A., Hogan, M. E., & Harmon-Jones, C. (2002). Proneness to hypomania/mania or depression and asymmetrical frontal cortical responses to an anger-evoking event. *Journal of Personality and Social Psychology, 82*, 610–618.

Hops, H., Biglan, A., Tolman, A., Arthur, J., & Longoria, N. (1995). *Living in Family Environments (LIFE) coding system: Manual for coders (Revised)*. Eugene, OR: Oregon Research Institute.

Ingram, R. E., Trenary, L., Odom, M., Berry, L., & Nelson, T. (2007). Cognitive, affective and social mechanisms in depression risk: cognition, hostility, and coping style. *Cognition & Emotion, 21*, 78–94.

Joiner, T. E. (2000). Depression's vicious scree: self-propagating and erosive processes in depression chronicity. *Clinical Psychology: Science and Practice, 7*, 203–218.

Katz, L. F., & Hunter, E. C. (2007). Maternal meta-emotion philosophy and adolescent depressive symptomatology. *Social Development, 16*, 343–360.

Kobak, R. R., Cole, H. E., Ferenz-Gillies, R., & Fleming, W. S. (1993). Attachment and emotion regulation during mother-teen problem solving: a control theory analysis. *Child Development, 64*, 231–245.

Kuppens, P., Allen, N. B., & Sheeber, L. (2010). Emotional inertia and psychological maladjustment. *Psychological Science, 21*, 984–991.

Levenson, R. W. (1992). Autonomic nervous system differences among emotions. *Psychological Science, 3*, 23–27.

Luutonen, S. (2007). Anger and depression—theoretical and clinical considerations. *Nordic Journal of Psychiatry, 61*, 246–251.

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of family context in the development of emotion regulation. *Social Development, 16*, 361–388.

Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry, 57*, 14–20.

Newman, J. L., Gray, E. A., & Fuqua, D. R. (1999). Sex differences in the relationship of anger and depression: an empirical study. *Journal of Counselling and Development, 77*, 198–203.

Orvaschel, H., & Puig-Antich, J. (1994). *Schedule for affective disorders and schizophrenia for school-age children-epidemiologic version-5 (K-SADS-E-5)*. Ft. Lauderdale, Fl: Nova Southeastern University.

Painuly, N., Sharan, P., & Mattoo, S. K. (2007). Antecedents, concomitants and consequences of anger attacks in depression. *Psychiatry Research, 153*, 39–45.

Parke, R. D., & McDowell, D. J. (1998). Towards an expanded model of emotion socialization: new people, new pathways. *Psychological Inquiry, 9*, 303–307.

Phares, V. (1992). Where's Poppa? The relative lack of attention to the role of fathers in child and adolescent psychopathology. *American Psychologist, 47*, 656–664.

Price, J. S., & Solomon, L. (1987). Depression as yielding behaviour: an animal model based on Schjelderup–Ebb's pecking order. *Ethology and Sociobiology, 8*, 85–98.

Prinz, R. J., Foster, S., Kent, R. N., & O'Leary, K. D. (1979). Multivariate assessment of conflict in distressed and nondistressed mother–adolescent dyads. *Journal of Applied Behavior Analysis, 12*, 691–700.

Radloff, L. S. (1977). A CES-D scale: a self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385–401.

Richards, J. E., & Casey, B. J. (1992). Development of sustained visual attention in the human infant. In B. A. Campbell, H. Hayne, & R. Richardson (Eds.), *Attention and information processing in infants and adults* (pp. 30–61). Hillsdale, NJ: Lawrence Erlbaum.

Roberts, R. E., Andrews, J. A., Lewinsohn, P. M., & Hops, H. (1990). Assessment of depression in adolescents using the center for epidemiologic studies depression scale. *Psychological Assessment, 2*, 122–128.

Rottenberg, J., Gross, J. J., & Gotlib, I. H. (2005). Emotion context insensitivity in major depressive disorder. *Journal of Abnormal Psychology., 114*, 627–639.

Severson, H. H., & Ary, D. V. (1983). Sampling bias due to consent procedures with adolescents. *Addictive Behaviors, 8*, 433–437.

Sheeber, L., Allen, N., Davis, B., & Sorensen, E. (2000). Regulation of negative affect during mother-child problem-solving interactions: adolescent depressive status and family processes. *Journal of Abnormal Child Psychology, 28*, 467–479.

Sheeber, L. B., Davis, B., Leve, C., Hops, H., & Tidesley, E. (2007). Adolescents' relationships with their mothers and fathers: associations with depressive disorder and subdiagnostic symptomatology. *Journal of Abnormal Psychology, 116*, 144–154.

Sheeber, L., Allen, N. B., Leve, C., Davis, B., Shortt, J. W., & Katz, L. F. (2009). Dynamics of affective experience and behavior in depressed adolescents. *Journal of Child Psychology and Psychiatry, 50*, 1419–1427.

Sigmon, S. T., & Nelson-Gray, R. O. (1992). Sensitivity to aversive events in depression: antecedent, concomitant, or consequent? *Journal of Psychopathology and Behavioural Assessment, 14*, 225–246.

Snijders, T. A. B., & Bosker, R. J. (1999). *Multilevel analysis: An introduction to basic and advanced multilevel modeling*. London: Sage.

Turpin, G. (1986). Effects of stimulus intensity on autonomic responding: the problem of differentiating orienting and defense reflexes. *Psychophysiology, 23*, 1–14.

Wenze, S. J., Gunthert, K. C., & Forand, N. R. (2006). Influence of dysphoria on positive and negative cognitive reactivity to daily mood fluctuations. *Behaviour Research and Therapy, 45*, 915–927.